Atlas of Genetics and Cytogenetics
in Oncology and Haematology

OPEN ACCESS JOURNAL

INIST-CNRS

Gene Section
Review

TRIM27 (tripartite motif containing 27)

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Published in Atlas Database: July 2014

Online updated version : http://AtlasGeneticsOncology.org/Genes/TRIM27ID42092ch6p22.html
DOI: 10.4267/2042/56441

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Abstract

Review on TRIM27, with data on DNA/RNA, on the protein encoded and where the gene is implicated.

Identity

Other names: RFP, RNF76

HGNC (Hugo): TRIM27

Location: 6p22.1

DNA/RNA

Description

The unprocessed TRIM27 transcript spans 20.990 kb and is organised in 8 exons.
Reporter construct studies suggest the presence of elements between -365 and -68 relative to the transcription start point to be sufficient to drive basal gene transcription.
This promoter region is GC rich and has no typical TATA and CAAT boxes (Iwata et al., 1999).

Transcription

Alternative splicing of the human locus may generate 7 or more processed alternative transcripts. Three of these transcripts contain open reading frames and presumably give rise to protein products (Ensembl).
Transcript 1: 2969 bps (transcript ID ENST00000377199, protein encoding, protein ID ENSP00000366404);
Transcript 2: 2704 bps (transcript ID ENST00000377194, protein encoding, protein ID ENSP00000366399);
Transcript 3: 1813 bps (transcript ID ENST00000414543, protein encoding, protein ID ENSP00000400058);
Transcript 4: 7006 bps (transcript ID ENST00000481474, processed transcript without open reading frame);
Transcript 5: 687 bps (transcript ID ENST00000496091, processed transcript without open reading frame);
Transcript 6: 541 bps (transcript ID ENST00000467742, processed transcripts without open reading frame);
Transcript 7: 7006 bps (transcript ID ENST00000481474, retains intronic sequence relative to other coding transcripts, without open reading frame).

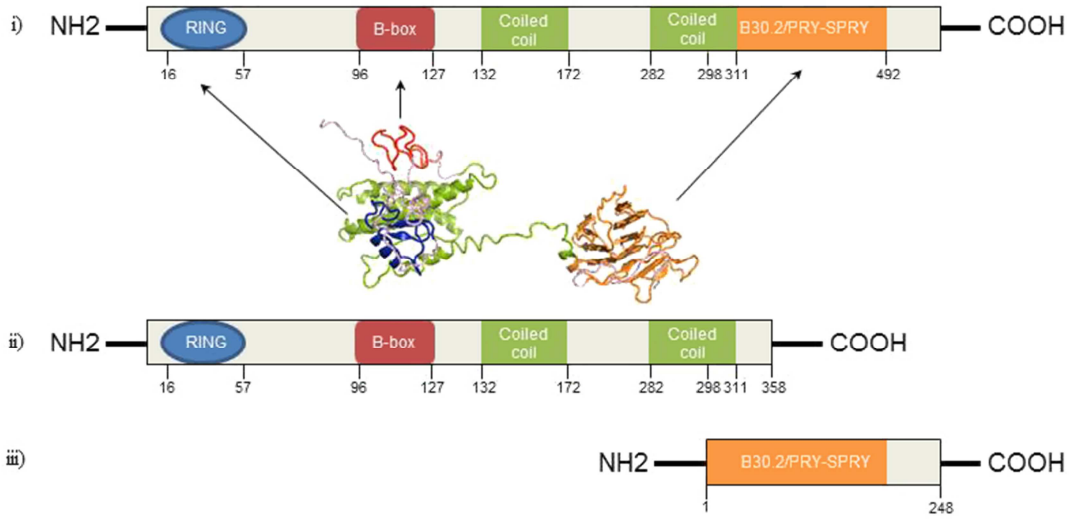
Pseudogene

There are no reported pseudogenes of TRIM27.

Protein

Description

The longest isoform 1 of TRIM27 (protein ID ENSP00000366404) encodes a 513 amino acids protein with a molecular weight of 58 KDa; displaying the so-called tripartite/RBCC motif within its N-terminal region, composed of a RING type zinc finger (InterPro ID IPR001841) (aa 16-57), a B-box type zinc finger (InterPro ID001841) (aa 96-127) and coiled-coil domains (aa 132-172 and aa 282-311), and a B30.2/PRY-SPRY domain (InterPro ID IPR001870) within its C-terminal region (aa 298-492).



Schematic diagram of TRIM27 protein. The domain structure of the three TRIM27 protein isoforms (i-iii) and a ribbon diagram of the structure of TRIM27 domains which represents a high confidence model generated by Phyre2 (http://www.sbg.bio.ic.ac.uk/phyre2/html/page.cgi?id=index). NH2, Amino-terminal region; COOH, Carboxyl-terminal region.

The RING finger may be involved in mediating protein-protein interactions and may confer ubiquitin family ligase activity. TRIM27 can form homo and hetero-oligomeric assemblies mediated by the centrally located coiled-coil domain (Cao et al., 1997). Isoform 2 (protein ID ENSP00000366399) lacks the C-terminal SPRY domain. Isoform 3 (protein ID ENSP00000400058) encodes the B30.2/PRY-SPRY domain only. The diagram above is a scheme of the protein isoforms and their domains.

Expression

TRIM27 transcripts and protein is ubiquitous in day 10.5-13.5 mouse embryos. Expression is restricted in adult mice, with the highest level of expression in pachytene spermatocytes and round spermatids of differentiating sperm (Cao et al., 1996; Tezel et al., 1999). TRIM27 is also expressed in mouse spleen, thymus, cerebrum and cerebellum. TRIM27 is expressed in various human and rodent cell lines. Based on RNAseq., TRIM27 mRNA is detected widely in human tissues, with no evidence for absence. Expression levels between tissues are similar, including testes, with comparative expression rating of low to medium across tissues (Human Protein Atlas).

Localisation

The protein localizes to the nucleus, cytoplasm and in nuclear bodies (NB) associated with the nuclear matrix (Isomura et al., 1992). Nuclear export of RFP is dependent on a functional NES, which can be activated by protein kinase C (PKC) (Harbers et al., 2001). Hetero-oligomerization has been observed between TRIM27 and TRIM19 with the recruitment of TRIM27 to the promyelocytic leukaemia nuclear bodies (PMLs) (Cao et al., 1998). Furthermore, interaction with members of the protein inhibitors of activated STAT (PIAS) family targets TRIM27 to subnuclear compartments/NBs, involving TRIM27 sumoylation (Matsuura et al., 2005). Nuclear or cytoplasmic localisation depends on the cell type or tissue.

Function

A role for TRIM27 has been reported in transcriptional regulation through interaction with retinoblastoma protein (RB1) and the Mi-2b-containing histone deacetylase complex in the nucleus (Krutzfeldt et al., 2005; Shimono et al., 2005), in transcriptional repression through interaction with the enhancer of polycomb protein (EPC) (Shimono et al., 2000; Bloor et al., 2005) and MBD proteins (Fukushige et al., 2006), in the negative regulation of NF-kB and IFN-signaling pathways (Zha et al., 2006), in the positive regulation of apoptosis through activation of Jun N-terminal kinase (JNK) (Dho and Kwon, 2003) and augmentation of TNF alpha receptor activation (Zaman et al., 2013), and in cell cycle regulation (Patel and Ghiselli, 2005) suggesting that TRIM27 is involved in the control of multiple cellular processes. TRIM27 has been shown to confer E3 ubiquitin ligase activity with the enzymes UBE2D1 and UBE2D3 (Napolitano et al., 2011) and to possess SUMO E3 ligase activity (Chu and Yang, 2011). TRIM27 regulates innate immune responses by physical interaction with NOD2. So, TRIM27 mediates K48-linked ubiquitination and subsequent proteasomal degradation of NOD2 (Zurek et al., 2012). TRIM27 has a critical role in regulating PTEN phosphatase activity through ubiquitination which inhibits PTEN increasing AKT signalling (Lee et al., 2013). TRIM27 functions as an E3

ligase to ubiquitinate and inhibit PI3K-C2beta's kinase activity and negatively regulates CD4 T cells (Cai et al., 2011).
TRIM27 also negatively regulates IgE receptor activation and downstream signalling by the same mechanism (Srivastava et al., 2012). TRIM27 interacts with MAGE-L2 protein and ubiquitination of WASH K220 by MAGE-L2-TRIM27 is required for endosomal F-actin nucleation and retrograde transport (Hao et al., 2013). MRTF-B/TRIM27 complex plays a role in the regulation of integrin b1 expression via Rho and miR-124 in Leading Cells (LCs) among migrating cancer cell groups following loss of intracellular adhension (Kato et al., 2014).

Homology

H. sapiens, TRIM27 tripartite motif containing 27, 513 aa.
P. troglodytes (chimpanzee), TRIM27 tripartite motif containing 27, 513 aa (XP_001140726.2).
M. mulatta, TRIM27 tripartite motif containing 27, 513 aa (XP_001094465.2).
C. lupus, TRIM27 tripartite motif containing 27, 487 aa (XP_005642504.1).
B. taurus, TRIM27 tripartite motif containing 27, 513 aa (XP_001069267.1).
M. musculus, Trim27 tripartite motif containing 27, 513 aa (NP_033080.2).
R. norvegicus, Trim27 tripartite motif containing 27, 513 aa (NP_001128446.1).
G. gallus, TRIM27 tripartite motif containing 27, 505 aa (NP_001092824.1).
With other RING finger/B box proteins.

Mutations

Germinal

No germinal mutations described for TRIM27.

Somatic

TRIM27 have been identified in cancer tissues by large scale tumour genome sequencing at low frequency (see table above). COSMIC lists 26 mutations from sequence analysis of 8960 cancers (mutation rate 0.29%).
Mutation spectrum: one nonrecurrent, 8 synonymous substitutions, one nonsense mutation, 16 nonsynonymous missense substitutions mostly leading to conservative aminoacid changes. Evidence is not available at present whether these observed mutations affect function of TRIM27 or confer selective advantage to tumour cells (COSMIC).

Tissue	Histology/Type	cDNA Mutation	Protein	Mutation	Reference
Bladder	NS	c.126C>T	p.R42R	Substitution-coding silent	Forbes et al., 2011
Bladder	NS	c.499G>A	p.A167T	Substitution- missense	Forbes et al., 2011
Colon	Adenocarcinoma	c.27C>T	p.C9C	Substitution-coding silent	Forbes et al., 2011
Colon	Adenocarcinoma	c.1269A>G	p.P423P	Substitution-coding silent	Forbes et al., 2011
Rectum	Adenocarcinoma	c.798T>A	p.P266P	Substitution-coding silent	Muzny et al., 2012
Endometrium	Endometrioid carcinoma	c.1505T>C	p.V502A	Substitution- missense	Forbes et al., 2011
Endometrium	Endometrioid carcinoma	c.503G>A	p.R168Q	Substitution- missense	Forbes et al., 2011
Endometrium	Endometrioid carcinoma	c.543G>A	p.K181K	Substitution-coding silent	Forbes et al., 2011
Kidney	Clear cell renal carcinoma	c.1128G>T	p.E376D	Substitution- missense	Sato et al., 2013
Liver	NS	c.862A>G	p.T288A	Substitution- missense	Forbes et al., 2011
Liver	Hepatocellular carcinoma	c.927A>G	p.R309R	Substitution-coding silent	Forbes et al., 2011
Lung	Adenocarcinoma	c.754_755GG>TT	p.G252L	Substitution-missense	Forbes et al., 2011
Lung	Adenocarcinoma	c.801G>T	p.W267C	Substitution-missense	Forbes et al., 2011
Lung	Adenocarcinoma	c.1205C>G	p.P402R	Substitution-missense	Forbes et al., 2011
Lung	Adenocarcinoma	c.1078G>T	p.V360F	Substitution-missense	Forbes et al., 2011
Lung	Small cell carcinoma	c.658A>G	p.T220A	Substitution-missense	Peifer et al., 2012
Lung	Small cell carcinoma	c.901G>C	p.D301H	Substitution-missense	Peifer et al., 2012
Lung	Adenocarcinoma	c.1481G>T	p.G494V	Substitution-missense	Imielinski et al., 2012
Lung	Adenocarcinoma	c.291G>C	p.E97D	Substitution-missense	Imielinski et al., 2012
Lung	Adenocarcinoma	c.1068G>T	p.L356L	Substitution- coding silent	Forbes et al., 2011
Lung	Adenocarcinoma	c.168C>G	p.C56W	Substitution- missense	Forbes et al., 2011
Lung	Adenocarcinoma	c.960G>C	p.L320L	Substitution- coding silent	Forbes et al., 2011
Lung	Adenocarcinoma	c.1302G>T	p.Q434H	Substitution- missense	Forbes et al., 2011
Lung	Adenocarcinoma	c.738G>T	p.E246D	Substitution- missense	Forbes et al., 2011
Ovary	Serous carcinoma	c.1024delT	p.Y342fs*34	Deletion-frameshift	Bell et al., 2011
Oesophagus	Adenocarcinoma	c.318C>A	p.Y106*	Substitution- nonsense	Dulak et al., 2013

TRIM27 mutations identified in cancer.

Implicated in

Thyroid papillary carcinoma (TPC)

Disease

A chromosomal translocation involving TRIM27/RFP is found in human thyroid papillary carcinomas.

TPC is the most common cancer of the thyroid gland that usually begins as a small irregular solid lump (nodule) in the thyroid gland.

Classical papillary carcinoma is characterised by the formation of papillae and a set of distinctive nuclear features (optically clear appearance, overlapping, pseudo-inclusions and nuclear grooves) (Rosai et al., 1992).

Cytogenetics

t(6;10)(p21.3;q11.2)

Hybrid/Mutated gene

TRIM27-RET; deltaTRIM27-RET.

Abnormal protein

Fusion of the N-terminus tripartite motif of TRIM27 with the truncated C-terminus tyrosine kinase domain of the c-ret proto-oncogene result in production of the ret transforming protein (Hasegawa et al., 1996; Saenko et al., 2003).

The level of catalytic activity of the hybrid TRIM27-RET protein is higher than that of RET alone (Kato et al., 2000).

Lung cancer

Note

TRIM27 mRNA or protein is highly expressed in human lung cancers.

In a study that uses a cancer-profiling array containing tumour cRNA alongside cRNA from paired tissue, TRIM27 expression was found to be significantly increased in lung cancers versus normal tissue (Zoumpoulidou et al., 2012). TRIM27 expression is associated with poor prognosis of lung cancers with EGFR mutations suggesting that TRIM27 status might be associated with response to anticancer therapy in EGFR-mutated lung cancers (Iwakoshi et al., 2012).

Endometrial cancer

Note

Positive TRIM27 expression in human endometrial cancer cells is correlated with poor clinical outcome in patients (Tsukamoto et al., 2009). Immunohistochemical analysis of TRIM27 expression in non-neoplastic endometrium samples as well as type I endometrial cancer in comparison to type II endometrial cancer has shown that TRIM27 positive expression was strongly correlated with serous carcinoma morphology (Tezel et al., 2012).

Breast cancer

Note

Analysis of TRIM27 expression by Townson et al. revealed TRIM27 is expressed in breast cancer derived cell lines regardless of estrogen receptor (ER) status. However, it is not clear to what extend TRIM27 is involved in breast tumorigenesis (Townson et al., 2006). In a tissue microarray (TMA) study, TRIM27 protein expression was found in breast carcinomas but not in benign breast tissues. TRIM27 expression also correlates with ERBB2 protein expression and ERBB2 gene amplification, which is a marker for poor prognosis in breast cancer (Tezel et al., 2009). However, the biological significance of this correlation is not known.

Ovarian cancer

Note

Immunohistochemical analysis of TRIM27 expression in epithelial ovarian cancer shows that TRIM27 expression significantly correlates with chemoresistance in ovarian cancer patients. In addition, positive TRIM27 staining was increased in recurrent ovarian cancer, correlating with a poor outcome of the patients. TRIM27 protein expression was significantly higher in platinum-resistant than in platinum-sensitive patients (Horio et al., 2012). Recombinant TRIM27 expression enhances chemoresistance of ovarian cancer cells to carboplatin and paclitaxel in culture and xenografts (Horio et al., 2012).

Murine squamous carcinoma of the skin

Note

TRIM27 is highly expressed in chemically induced squamous cell carcinomas (SCC) in the mouse after exposure of the skin to the carcinogen 7, 12-dimethylbenzanthracene (DMBA)/12-O-tetradecanoylphorbol-13-acetate (TPA), as part of a two-step carcinogenesis model. Trim27 expression peaks in early mouse cancer development (Zoumpoulidou et al., 2012). Moreover, mice with TRIM27 deletion are resistant to development of chemically induced SSC (Zoumpoulidou et al., 2012).

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This article should be referenced as such:

Zoumpoulidou G, Mittnacht S. TRIM27 (tripartite motif containing 27). Atlas Genet Cytogenet Oncol Haematol. 2015; 19(4):302-307.